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SEA KATAYAMA, T?/AU

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     ESBIOBASE' ENTERED AT 09:54:15 ON 10 OCT 2003
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L4             2 DUP REM L3 (10 DUPLICATES REMOVED)

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9 ANSWER 40 OF 47 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
AN 97:116910 SCISEARCH
GA The Genuine Article (R) Number: WE836
TI **Fibronectin** suppresses **apoptosis** in normal human melanocytes through an integrin-dependent mechanisms
AU Scott G (Reprint); Cassidy L; Busacco A
CS UNIV ROCHESTER, SCH MED & DENT, DEPT DERMATOL, 601 ELMWOOD AVE, ROCHESTER, NY 14642 (Reprint); UNIV ROCHESTER, SCH MED & DENT, DEPT PATHOL, ROCHESTER, NY 14642
CYA USA
SO JOURNAL OF INVESTIGATIVE DERMATOLOGY, (FEB 1997) Vol. 108, No. 2, pp. 147-153.
Publisher: BLACKWELL SCIENCE INC, 238 MAIN ST, CAMBRIDGE, MA 02142.
ISSN: 0022-202X.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 44
*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
AB Recent reports show that components of the extracellular matrix function as cell survival factors through the suppression of apoptosis (programmed cell death). In this report we show that attachment to **fibronectin** suppresses **apoptosis** of normal human fetal and neonatal melanocytes in vitro and that prevention of attachment to underlying matrix or attachment to poly-l-lysine is a potent inducer of apoptosis in melanocytes. A role for the beta 1-integrin family in mediating cell survival signals was shown by the ability of beta 1-blocking antibodies to enhance apoptosis in melanocytes attached to **fibronectin**, and by the ability of anti-beta 1 antibodies immobilized on solid supports to suppress **apoptosis** in melanocytes. Cytochalasin D reversed the effect of **fibronectin** on the suppression of **apoptosis** in melanocytes, suggesting that an intact cytoskeleton is required for transduction of survival signals. A human metastatic melanoma cell line, SKMEL28, was resistant to apoptosis when grown in suspension or on poly-l-lysine, even after 4 d in culture in the absence of exogenous growth factors. These results suggest that **fibronectin** suppresses **apoptosis** in normal human melanocytes through an integrin-dependent pathway and that significant differences in the control of anchorage-dependent regulation of apoptosis exist in melanocytes and melanoma cells.

L9 ANSWER 41 OF 47 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
AN 97:102327 SCISEARCH
GA The Genuine Article (R) Number: WE221
TI Differential effect of components of the extracellular matrix on differentiation and apoptosis
AU Aharoni D; Meiri I; Atzmon R; Vlodavsky I; Amsterdam A (Reprint)
CS WEIZMANN INST SCI, DEPT MOL CELL BIOL, IL-76100 REHOVOT, ISRAEL (Reprint); WEIZMANN INST SCI, DEPT MOL CELL BIOL, IL-76100 REHOVOT, ISRAEL; HEBREW UNIV JERUSALEM, HADASSAH UNIV HOSP, DEPT ONCOL, IL-91120 JERUSALEM, ISRAEL
CYA ISRAEL

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ANSWER 13 OF 16 MEDLINE on STN DUPLICATE 8
AN 1998341321 MEDLINE
DN 98341321 PubMed ID: 9665806
TI Modulation of apoptotic cell death by extracellular matrix proteins and a fibronectin-derived antiadhesive peptide.
AU Fukai F; Mashimo M; Akiyama K; Goto T; Tanuma S; Katayama T
CS Department of Patho-Physiology, Faculty of Pharmaceutical Sciences, Science University of Tokyo, Tsukuba City, Japan.. fukai@ps.kagu.sut.ac.jp
SO EXPERIMENTAL CELL RESEARCH, (1998 Jul 10) 242 (1) 92-9.
Journal code: 0373226. ISSN: 0014-4827.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980820
Last Updated on STN: 19980820
Entered Medline: 19980810
AB Cell adhesion to the extracellular matrix (ECM) has been implicated in apoptosis in anchorage-dependent cell types. We recently found that a peptide derived from fibronectin (termed III14-2) inhibits the integrin-mediated cell adhesion to ECM. Using this antiadhesive peptide and a variety of ECM proteins, we show here a critical role of the integrin-ECM protein interaction in apoptotic regulation of human umbilical vein **endothelial** cells (HUVEC). HUVEC in suspension underwent apoptosis under the serum-free conditions, as judged by nuclear and DNA fragmentations. This apoptosis was suppressed to varying degrees when alpha 5 beta 1, alpha v beta 3, and alpha 2 beta 1 integrins were occupied with either soluble or immobilized ECM proteins such as fibronectin, vitronectin, and type I collagen, respectively. Peptide III14-2, which had no effect by itself on the HUVEC apoptosis, disrupted the ligation of alpha 5 beta 1 and alpha v beta 3 but no alpha 2 beta 1 and ultimately led the cells to apoptosis, indicating that this antiadhesive peptide indirectly induces apoptosis by blocking cell survival signal delivered from alpha 5 beta 1 and alpha v beta 3 integrins. Genistein, a protein tyrosine kinase inhibitor, slightly reduced the rescuing effect of **fibronectin**, whereas sodium orthovanadate and bombesin, which increase in the level of protein tyrosine phosphorylation, made HUVEC less susceptible to **apoptosis** and blocked the effect of peptide III14-2. HUVEC adhesion to fibronectin substrate raised the tyrosine phosphorylation level of focal adhesion kinase and the expression of cytoprotective Bcl-2 protein, both of which were reversed by the antiadhesive effect of peptide III14-2. Thus, the opposing effects of ECM proteins, including **fibronectin** and vitronectin, and peptide III14-2 on HUVEC **apoptosis** appear to be due to the opposing effects of these factors on the signaling pathway which includes tyrosine phosphorylation of FAK and Bcl-2 expression.

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apoptosis in these cells. On the basis of these observations, we hypothesized that signals triggered in neutrophils via their adhesion receptors might also modify their life span. This hypothesis has been tested using human neutrophils adherent to tissue culture plastic, either untreated or coated with extracellular matrix (ECM) proteins or with monolayers of human umbilical vein **endothelial** cells. To detect and quantitate apoptotic changes in adherent cells, we developed a microtiter plate assay using a cell-permeable DNA-binding fluorescent dye, Hoechst 33342. Use of this assay demonstrated that 1) the number of apoptotic cells among neutrophils adherent to plastic after 6-20 h of incubation was significantly lower than that among neutrophils adherent to the ECM proteins **fibronectin** or laminin; 2) adhesion to interleukin-1-activated **endothelial** cells delayed **apoptosis**, whereas adhesion to nonactivated endothelium accelerated neutrophil death; and 3) monoclonal antibodies directed against intercellular adhesion molecule 1 or against the common beta-2-chain of the leukocyte integrins abolished the protective effect of interleukin-1-activated **endothelial** cells on apoptosis of adherent neutrophils. These results suggest that the life span of adherent neutrophils depends on the activating signals triggered by the surface of adhesion.

**Fibronectin suppresses apoptosis** in normal human melanocytes through an integrin-dependent mechanism.

AU Scott G; Cassidy L; Busacco A

CS Department of Dermatology, University of Rochester School of Medicine and Dentistry, New York 14642, USA.

NC AR-01882-01 (NIAMS)

SO JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1997 Feb) 108 (2) 147-53.
Journal code: 0426720. ISSN: 0022-202X.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS MEDLINE; Priority Journals

OS MEDLINE 97160906

EM 199702

ED Entered STN: 19970305
Last Updated on STN: 19970509

AB Recent reports show that components of the extracellular matrix function as cell survival factors through the **suppression** of **apoptosis** (programmed cell death). In this report we show that attachment to **fibronectin suppresses apoptosis** of normal human fetal and neonatal melanocytes in vitro and that prevention of attachment to underlying matrix or attachment to poly-L-lysine is a potent inducer of **apoptosis** in melanocytes. A role for the beta1-integrin family in mediating cell survival signals was shown by the ability of beta1-blocking antibodies to enhance **apoptosis** in melanocytes attached to **fibronectin**, and by the ability of anti-beta1 antibodies immobilized on solid supports to **suppress apoptosis** in melanocytes. Cytochalasin D reversed the effect of **fibronectin** on the **suppression** of **apoptosis** in melanocytes, suggesting that an intact cytoskeleton is required for transduction of survival signals. A human metastatic melanoma cell line, SKMEL28, was resistant to **apoptosis** when grown in suspension or on poly-L-lysine, even after 4 d in culture in the absence of exogenous growth factors. These results suggest that **fibronectin suppresses apoptosis** in normal human melanocytes through an integrin-dependent pathway and that significant differences in the control of anchorage-dependent regulation of **apoptosis** exist in melanocytes and melanoma cells.

L7 ANSWER 14 OF 18 CAPLUS COPYRIGHT 2003 ACS on STN

AN 1997:369083 CAPLUS

DN 127:48345

TI The role of fibronectin in proliferation and differentiation of megakaryocytic cells

AU Fukazawa, Motoharu

ANSWER 35 OF 47 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN DUPLICATE 10

AN 97:347535 SCISEARCH

GA The Genuine Article (R) Number: WW569

TI Thrombospondin 1 and type I repeat peptides of thrombospondin 1 specifically induce apoptosis of **endothelial** cells

AU Guo N H; Krutzsch H C; Inman J K; Roberts D D (Reprint)

CS BLDG 10, ROOM 2A33, 10 CTR DR MSC 1500, BETHESDA, MD 20892 (Reprint); NIAID, NIH, BETHESDA, MD 20892; NCI, PATHOL LAB, BETHESDA, MD 20892

CYA USA

SO CANCER RESEARCH, (1 MAY 1997) Vol. 57, No. 9, pp. 1735-1742. Publisher: AMER ASSOC CANCER RESEARCH, PUBLIC LEDGER BLDG, SUITE 816, 150 S. INDEPENDENCE MALL W., PHILADELPHIA, PA 19106. ISSN: 0008-5472.

DT Article; Journal

FS LIFE; CLIN

LA English

REC Reference Count: 48

*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

AB Thrombospondin 1 (TSP1) inhibits angiogenesis and modulates **endothelial** cell adhesion, motility, and growth. The antiproliferative activity of TSP1 is mimicked by synthetic peptides derived from the type I repeats of TSP1 that antagonize fibroblast growth factor 2 and activate latent transforming growth factor beta. These TSP1 analogues induced programmed cell death in bovine aortic **endothelial** cells based on morphological changes, assessment of DNA fragmentation, and internucleosomal DNA cleavage. Intact TSP1 also induced DNA fragmentation The **endothelial** cell response was specific because no DNA fragmentation was induced in MDA-MB-435S breast carcinoma cells, although TSP1 and the peptide conjugates inhibited the growth of both cell types. Apoptosis did not depend on activation of latent transforming growth factor beta because peptides lacking the activating sequence RFK were active. Apoptosis was not sensitive to inhibitors of ceramide generation but was inhibited by the phosphatase inhibitor vanadate. Induction of DNA fragmentation by the peptides nas decreased when **endothelial** cell cultures reached confluence. Growth of the cells on a **fibronectin** substrate also suppressed induction of **apoptosis** by TSP1 or the peptides. Differential sensitivities to kinase inhibitors suggest that apoptosis and inhibition of proliferation are mediated by distinct signal transduction pathways. These results demonstrate that induction of apoptosis by the TSP1 analogues is not a general cytotoxic effect and is conditional on a lack of strong survival-promoting signals, such as those provided by a **fibronectin** matrix. The antitumor activity of TSP1 may therefore result from an increased sensitivity to **apoptosis** in **endothelial** cells adjacent to a provisional matrix during formation of vascular beds in tumors expressing TSP1.

ANSWER 36 OF 47 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS, INC. on STN